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New Modalities for a New Generation: Casein Phosphopeptide-Amorphous Calcium Phosphate, A New Remineralisation Technology

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Abstract

Dental caries is the localised destruction of tooth tissue by specific dental plaque bacteria that ferment dietary sugar to organic acids. Even though in most developed countries the prevalence of dental caries has decreased through the use of fluorides, the disease remains a major public health problem. A substantial volume of literature now exists demonstrating an anticariogenic effect of dairy products [milk, milk concentrates, powders and cheeses]. This anticariogenic effect has been attributed to the multiphosphoseryl-containing sequences of casein. These sequences can be released as casein phosphopeptides (CPP) from an enzymatic digest of casein. The CPP have a remarkable ability to stabilise calcium phosphate in solution as amorphous calcium phosphate (ACP) nanocomplexes. Through their multiple phosphoseryl residues, the CPP bind to ACP in metastable solution preventing their growth to the critical size required for nucleation and phase transformation to an insoluble crystalline calcium phosphate. NMR conformational and binding studies have shown that CPP phosphoseryl residues are essential for ACP interaction. The casein phosphopeptide-amorphous calcium phosphate nanocomplexes complexes (CPP-ACP) have been shown to localise at the tooth surface and prevent enamel demineralisation in laboratory, animal and human *in situ* trials. The CPP-ACP have also been shown to remineralise enamel subsurface lesions *in situ* when delivered in oral care products. The proposed anticariogenic mechanism for CPP-ACP is the localisation of ACP at the tooth surface which buffers the free calcium and phosphate ion activities, thereby helping to maintain a state of supersaturation with respect to tooth enamel, preventing demineralisation and enhancing remineralisation. The CPP-ACP interact with fluoride ions to produce an amorphous calcium fluoride phosphate stabilised by the CPP at the tooth surface. This provides soluble calcium, fluoride and phosphate ions to promote remineralisation with fluorapatite that is more resistant to future acid challenge. Oral care products containing CPP-ACP (Recaldent™) are now commercially available in Australia, USA, Europe and Japan.

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THIS ISSUE

"Standards become valuable when they are well known, and easily accessible."

President's Report

Is it all just a waste of time then?

Are our efforts in vain when we attempt to repair the damage done by caries in the primary dentition? This is the challenge that has been thrown down to those of us who provide dental care for children. Milsom *et al*. (2002) found that the risk of a carious primary molar being extracted was similar whether the teeth had received restorative treatment or not. Fayle devoted a session at the ANZSPD Biennial Convention in March last year, to this very question. His lucid presentation examined the available evidence and was the catalyst for much valuable discussion amongst the delegates. Broadly speaking, Fayle's conclusion was that appropriately chosen and well-implemented restorative dental treatment was effective. Not surprisingly, he suggested that it is not the amount of treatment, but the standard of care, which determines the effectiveness of our interventions.

Our organisation, the Australian and New Zealand Society of Paediatric Dentistry (ANZSPD), is committed to the study and advancement of paediatric dentistry, and the promotion of education in the field of paediatric dentistry. Some of our members may not be aware that since 1992, a smaller organisation for specialists in the field of paediatric dentistry, which has broadly similar aims to ANZSPD, has developed from within our ranks to form the independent Australasian Academy of Paediatric Dentistry (AAPD). The AAPD was not formed to hijack the agenda for paediatric dentistry, but to provide a necessary professional organisation to represent those who have chosen to become specialists in the field of paediatric dentistry. As a member of both the ANZSPD and the AAPD, I feel that one of the most important clauses in the AAPD constitution is the one requiring AAPD members to remain active members of their local Society of Paediatric Dentistry.

In the Synopses of June 2003, our immediate past President, Dr Chris Olsen referred to a major project of the AAPD, the *Standards of Care* in Paediatric Dentistry, which was at the time being completed and ratified by the members of the Academy. The *Standards of Care* is a significant document and is the result of an enormous effort by the members of the Academy who reviewed the available international guidelines and standards of paediatric dental care, together with the published scientific literature to distill and localise these guidelines for Australia and New Zealand. Until now, the *Standards of Care* have been confined to the 'members-only' area of the AAPD website.

Standards become valuable when they are well known, and easily accessible, so I am pleased to report that the current President of AAPD, Dr Peter Wong has agreed to make the *Standards of Care* available to all members of ANZSPD in the near future via a link from our website (www.anzspd.org.au). The dissemination of the Academy's *Standards of Care* to ANZSPD reflects the reality of our profession, that the vast majority of paediatric dental care in our region is carried out by non-specialist dental practitioners. The Academy's *Standards of Care* represent a major step forward in the promotion of excellence in the delivery of dental care to children and adolescents in our region and should be a great resource for ANZSPD members.

Preparations are now well in hand for the 20th IAPD Congress in 2005 which is being hosted by ANZSPD. The Chairman of the Organising Committee, Dr Richard Widmer, reports strong support for the IAPD Congress, and he has promised a stimulating and rewarding scientific and social program. If you haven't already done so, visit the website (www.iapd2005.com) and register your interest. Please mark your diaries now so that you will join us all in Sydney from 31 October to 5 November 2005.

While you are doing your forward planning, the next Biennial Conference of ANZSPD will be held in Broome, Western Australia at the Cable Beach Resort in May 2007.

John Winters

The *Standards of Care* document

A number of years ago the Australasian Academy of Paediatric Dentistry decided to produce a '*Standards of Care*' (SOC) document. As the organisation representing specialist paediatric dentists, it was considered important that we produce a series of guidelines and recommendations for the management of all facets of paediatric dental care in children.

Under the guiding force of our past president Dr Peter Gregory, the members of our academy spent many long hours and meetings, developing a series of guidelines and recommendations which encompass the 'SOC'. The guidelines in this document attempt to combine all facets of paediatric dental care. The document defines and lists recommendations for restorative care, hospital dentistry, radiography hygiene, special care dentistry, dental sedation, and all significant areas of dental care in children.

The SOC is not considered to be a textbook rather a document that can be referred to when advice or recommendations are required.

The document has been produced in hard copy and has been distributed to all of the Dental Boards within Australia. It has also been sent to the federal ADA and the local ADA branches within Australia.

The executive of the AAPD with the coordination of Dr Eduardo Alcaino are presently taking steps to make the SOC available on the ADA, NZDA and ANZSPD web sites. This has proven to be a slow process but be assured that it is underway.

Finally, I do have some hard copies of the SOC available (up to 20 only), and if any ANZSPD members would like a copy (first 20 please) feel free to give me a call or email me on pwpedo@alphalink.com.au.

Dr Peter Wong
President, AAPD

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Dental caries

Dental caries is initiated via the demineralisation of tooth hard tissue by organic acids from the fermentation of dietary sugar by dental plaque odontopathogenic bacteria[1]. Even though in most developed countries the prevalence of dental caries has decreased through the use of fluorides, the disease remains a major public health problem[2]. In a recently published 1996 Australian child dental health survey 40.2% of 6 year olds and 48.6% of 12 year olds showed signs of dental caries[2]. Untreated decay in the combined deciduous and permanent dentition was present in 35.3% of children in the age range 5 to 15 years with the greatest severity occurring in the youngest ages (eg. 9.1% of 5 year olds had 4 or more teeth with untreated decay). The level of disease in these high-risk children has decreased only slightly in recent years (eg. the proportion of 6 year olds with 4 or more caries affected teeth reduced by only 3.7% between 1989 and 1996). Recent dental health surveys in adolescents and adults have indicated that the gains in oral health made in childhood are not necessarily carried into later years as these age groups exhibit higher percentages of high caries-risk individuals. For example, 78% of young adults selected from the electoral roll in Adelaide showed signs of caries with a mean DMFT of 3.66 and over 10% of these individuals exhibited a DMFT of 8 or more[2]. Demographic changes and changing patterns of oral disease are resulting in larger numbers of older Australians being dentate. A recent survey of nursing home residents showed that 34% of the residents were dentate with 41% of their teeth showing signs of decay and a mean DMFT of 23.7.

The estimated economic burden of treating dental caries in most developed communities is higher than that for other dietary related diseases including coronary heart disease, hypertension or stroke[3]. Recent studies have highlighted a number of socio-demographic variables associated with caries risk; high risk being associated with ethnicity and low socio-economic status[4]. Dental caries, therefore is still a major public health problem in Australia, particularly in ethnic and lower socio-economic groups who tend not to use dental services.

During the caries process the organic acids produced by the plaque bacteria diffuse into the tooth enamel via the water-filled interprismatic spaces and dissolve apatite crystals in a process referred to as demineralisation. This loss of calcium phosphate from the enamel structure results in the development of an incipient subsurface enamel lesion. At this stage the caries process is reversible and it is possible for calcium and phosphate ions, particularly the neutral ion pair $CaHPO_4^0$, to diffuse into the subsurface lesion to restore the lost apatite in a process referred to as remineralisation. However, the use of remineralisation solutions containing calcium and phosphate ions clinically has not been successful due to the low solubility of calcium phosphates, particularly in the presence of fluoride ions. Insoluble calcium phosphates are not easily applied, do not localise effectively at the tooth surface and require acid for solubility. Soluble calcium phosphate ions are at low concentrations, and also do not substantially incorporate into plaque or localise at the tooth surface. A new technology has now emerged involving an amorphous form of calcium phosphate stabilised by phosphopeptides from the milk protein casein.

Anticariogenic casein phosphopeptides

The food group most recognised as exhibiting anticaries activity is dairy products [milk, milk concentrates, powders and cheeses)[5]. Using *in vitro*, animal and *in situ* caries models, the components largely responsible for this anticariogenic activity have been identified as casein, calcium and phosphate[5-11]. The bovine milk phosphoprotein, casein, which is known to interact with calcium and phosphate[12] and is a natural food component is an obvious candidate for an anticariogenic food and toothpaste additive, however this is precluded by organoleptic properties and the very high levels required for activity[5]. Using a human intra-oral caries model[10] it has been shown that digestion of caseinate with trypsin did not destroy the protein's ability to prevent enamel sub-surface demineralisation. Tryptic peptides of casein were found incorporated into the intra-oral appliance plaque and were associated with a substantial increase in the plaque's content of calcium and phosphate. It was concluded that the tryptic peptides that were responsible for the anticariogenic activity were the calcium phosphate sequestering phosphopeptides. The casein phosphopeptides (CPP) released by trypsin that sequester calcium phosphate are Bosα_{s1}-casein X-5P (f59-79) **[1]**, Bosβ-casein X-4P (f1-25) **[2]**, Bosα_{s2}-casein X-4P (f46-70) **[3]** and Bosα_{s2}-casein X-4P (f1-21) **[4]** using Ser*(P)* to represent a phosphoseryl residue.

[1] Gln^{59}-Met-Glu-Ala-Glu-Ser*(P)*-Ile-Ser(P)-Ser*(P)*-Ser*(P)*-Glu-Glu-Ile-Val-Pro-Asn-Ser*(P)*-Val-Glu-Gln-Lys^{79}.α_{s1}(59-79).

[2] Arg^{1}-Glu-Leu-Glu-Glu-Leu-Asn-Val-Pro-Gly-Glu-Ile-Val-Glu-Ser*(P)*-Leu-Ser*(P)*-Ser*(P)*-Ser*(P)*-Glu-Glu-Ser-Ile-Thr-Arg^{25}.β(1-25).

[3] Asn^{46}-Ala-Asn-Glu-Glu-Glu-Tyr-Ser-Ile-Gly-Ser*(P)*-Ser*(P)*-Ser*(P)*-Glu-Glu-Ser*(P)*-Ala-Glu-Val-Ala-Thr-Glu-Glu-Val-Lys^{70}.α_{s2}(46-70).

[4] Lys^{1}-Asn-Thr-Met-Glu-His-Val-Ser*(P)*-Ser*(P)*-Ser*(P)*-Glu-Glu-Ser-Ile-Ile-Ser*(P)*-Ser*(P)*-Gln-Glu-Thr-Tyr-Lys^{21}.α_{s2}(1-21).

These peptides are 10% w/w of caseinate and through their multiple phosphoseryl residues sequester their own weight in calcium phosphate to form colloidal complexes[12,13]. As the CPP are not associated with the unpalatability[13] or allergenicity[14], of the caseins and have the potential for a specific anticariogenicity at least ten times greater on a weight basis, then their potential as a food and toothpaste additive is considerably better than that of the intact proteins. The CPP can be purified efficiently by microfiltration of calcium phosphate-induced complexes of the multiple phosphoseryl-containing peptides of a tryptic digest of casein[15]. The peptides produced by this procedure have been comprehensively characterised[16] and are β(1-25) **[2]** and α_{s1}(59-79) **[1]** and its deamidated forms with smaller amounts of α_{s2}(46-70) **[3]** and α_{s2}(1-21) **[4]** All peptides contain the sequence Ser*(P)*-Ser*(P)*-Ser*(P)*-Glu-Glu. The individual peptides of the preparation were identified by amino acid composition and sequence analyses after purification to homogeneity by anion exchange FPLC and reversed-phase HPLC[16]. Prior to sequence analysis the labile phosphoseryl residues were converted to *S*-ethyl cysteinyl residues by β-elimination[16].

Interaction of CPP with calcium phosphate

The CPP have a substantial ability to stabilise calcium phosphate in solution. Solutions containing 0.1% w/v α_{s1}(59-79) **[1]** at various pH, calcium and phosphate concentrations, but constant ionic strengths were used to characterise the peptide's interaction with calcium phosphate. The peptide was found to bind 21 Ca and 14 Pi per molecule. The ion activity products for the various calcium phosphate phases [hydroxyapatite (HA); octacalcium phosphate (OCP); tricalcium phosphate (TCP); amorphous calcium phosphate (ACP); and dicalcium phosphate dihydrate (DCPD) were determined from the free calcium and phosphate concentrations at each pH using a computer program that calculates the ion activity coefficients through the use of the expanded Debye-Hückel equation and takes into account the ion pairs $CaHPO_4^0$, $CaH_2PO_4^+$, $CaPO_4^-$ and $CaOH^+$ the dissociation of H_3PO_4 and H_2O and the ionic strength. The only ion activity product that significantly correlated with calcium phosphate bound to the peptide independently of pH was that corresponding to a basic ACP phase [$Ca_{3.0877}(PO_4)_2(OH)_{0.1754}xH_2O$] suggesting that this is the phase stabilised by α_{s1}(59 79). In neutral and alkaline supersaturated calcium phosphate solutions ACP nuclei spontaneously form. It is proposed that the peptide α_{s1}(59-79) binds to the forming ACP nanoclusters producing a metastable solution preventing ACP growth to the critical size required for nucleation and phase transformation. From stoichiometric analysis the stabilized nanoclusters had the unit formula [α_{s1}(59-79)$(ACP)_7]_n$ where n is equal to or greater than one. A 1.0% w/v CPP solution can stabilise 60 mM $CaCl_2$ and 36 mM sodium phosphate at pH 7.0 to form colloidal amorphous calcium phosphate-CPP nanocomplexes (CPP-ACP). This solution has been studied using a variety of *in vitro*, human *in situ* and animal caries models.

Anticariogenicity of CPP-ACP in the rat

The ability of casein-phosphopeptide amorphous calcium-phosphate nanocomplexes (CPP-ACP) to reduce caries activity was investigated using specific-pathogen-free rats orally infected with *Streptococcus sobrinus* 6715WT-13[17]. CPP-ACP solutions, applied to the animals teeth twice daily, significantly reduced caries activity with 0.1% w/v CPP-ACP producing a 14% reduction and 1.0% w/v CPP-ACP a 55% reduction relative to the distilled water control[17]. CPP-ACP at 0.5-1.0% w/v produced a reduction in caries activity similar to that of the 500 ppm F^- solution. The anticariogenicity of CPP-ACP and fluoride were additive as animals receiving 0.5% CPP-ACP plus 500 ppm F^- had significantly lower caries activity than those animals receiving either CPP-ACP or fluoride alone.

Anticariogenicity of CPP-ACP in human *in situ* studies

Enamel demineralisation *in situ*

The ability of the 1.0% CPP-ACP pH 7.0 solution to prevent enamel demineralisation has been studied in a human *in situ* caries model[18]. The model has been described in detail previously[10] and consists of a removable appliance containing a left and right pair of enamel slabs placed to produce a plaque retention site. The inter-enamel plaque that develops (3-5 mg) is bacteriologically similar to normal supragingival plaque[10]. On frequent exposure to sucrose solutions over a three week period the levels of mutans streptococci and lactobacilli increase and sub-surface enamel demineralisation results in the formation of a sub-surface enamel lesion. In this model two exposures of the CPP-ACP solution per day produced a 51%±19% reduction in enamel mineral loss relative to the control enamel. The plaque exposed to the CPP-ACP solution contained 78±22 µmol/g calcium, 52±25 µmol/g P_i and 242±71 µg/g CPP compared with 32±12 µmol/g calcium and 20±11 µmol/g Pi in the control plaque. The level of the CPP was determined by competitive ELISA using an antibody that recognised both α_{s1}(59-79) and β(1-25). Electron micrographs of immunocytochemically stained sections of the plaque revealed localisation of the peptide predominantly on the surface of microorganisms but also in the extracellular matrix. The incorporation of the CPP-ACP in the plaque resulted in a 2.4 fold increase in the plaque calcium and a 2.6 fold increase in plaque P_i with a Ca/P_i ratio consistent with ACP.

Enamel remineralisation *in situ*

The ability of CPP-ACP added to sugar-free chewing gum to remineralise enamel sub-surface lesions was investigated in a randomised, cross-over, double-blind clinical study[19]. Ten subjects wore removable palatal appliances with six, human-enamel, half-slabs inset containing sub-surface demineralised lesions. The other half of each enamel slab was stored in a humidified container and was used as the control demineralised lesion. There were three treatment groups in the study, sugar-free gum containing 0.6% w/w CPP-ACP, sugar-free gum not containing CPP-ACP and a nil-treatment control. The gums were chewed for 20 min periods, four times a day. The appliances were worn for this 20 min period and a further 20 min period after gum chewing. Each treatment was for 14 days duration and each of the ten subjects carried out each treatment with a one week rest between the treatments. At the completion of each treatment the enamel slabs were removed, paired with their respective demineralised control, embedded, sectioned and subjected to microradiography and computer-assisted densitometric image analysis to determine the level of remineralization. Chewing the sugar-free gum not containing CPP-ACP resulted in 9.16±1.16% remineralisation whereas the nil-treatment control effected 3.28±1.01% remineralisation. The gum containing 0.6% CPP-ACP however produced 18.35±3.02% enamel remineralisation, with all values being significantly different. SEM-EDAX analyses of sections of the remineralised enamel revealed that the mineral deposit was hydroxyapatite. These results showed that addition of 0.6% CPP-ACP to sugar-free gum produced a 100% increase in sub-surface enamel remineralisation. Microradiography of the enamel lesions before and after remineralisation showed that the CPP-ACP effected remineralization throughout the body of the lesion. Similar studies have now confirmed the ability of CPP-ACP in sugar-free gum to remineralise enamel subsurface lesions *in situ*[20,21]. A more

recent study has shown that enamel remineralised by CPP-ACP *in situ* was more resistant to further acid challenge that normal tooth enamel[22].

in vitro remineralisation of enamel lesions by CPP-ACP

Using an *in vitro* model system a number of solutions containing various amounts of CPP (0.1-1.0%), calcium (6-60 mM) and phosphate (3.6-36 mM) at different pH values (7.0-9.0) have been studied for their ability to remineralise artificial lesions in human third molar enamel[23]. The associations between the activities of the various calcium phosphate species in solution and the rate of enamel lesion remineralisation for this series of solutions were then determined. The activity of the neutral ion pair $CaHPO_4^O$ in the various remineralising solutions was found to be highly correlated with the rate of lesion remineralisation[23]. The diffusion coefficient for the remineralisation process was estimated at 3 x 10^{-10} m^2s^{-1} which is consistent with the coefficients of diffusion for neutral molecules through a charged matrix. $CaHPO_4^O$ and associated species after diffusion into the enamel lesion, would by the formation of Ca^{2+} and PO_4^{3-} ions, increase the degree of saturation with respect to HA. The formation of HA in the lesion would lead to the generation of acid and phosphate including H_3PO_4, which would diffuse out of the lesion down a concentration gradient. The results indicate that the CPP-bound ACP, acts as a reservoir of the neutral ion species $CaHPO_4^O$ that is formed in the presence of acid. The acid could be generated by dental plaque bacteria; under these conditions, the CPP-bound ACP would buffer plaque pH and produce calcium and phosphate ions, in particular $CaHPO_4^O$. The increase in plaque $CaHPO_4^O$ would offset any fall in pH thereby preventing enamel demineralisation. Acid is also generated in plaque as H_3PO_4 by the formation of HA in the enamel lesion during remineralisation. This therefore could explain why the CPP-ACP are such efficient remineralizing agents as they would consume the H_3PO_4 produced during enamel lesion remineralisation generating more $CaHPO_4^O$ thus maintaining its concentration gradient into the lesion. These results are therefore consistent with the proposed anticariogenic mechanism of the CPP being the inhibition of enamel demineralisation and enhancement of remineralisation through the localisation of ACP at the tooth surface.

Interaction of CPP-ACP with fluoride

The additive anticariogenic effect of the 1.0% CPP-ACP and 500 ppm F^- in the rat caries experiments[17] led to the investigation of potential interaction between the CPP-ACP and F. Analysis of the solution containing 1.0% CPP, 60 mM $CaCl_2$, 36 mM sodium phosphate and 500 ppm F (26.3 mM NaF) pH 7.0 after ultrafiltration revealed that nearly half of the fluoride ion had incorporated into the ACP phase stabilised by the CPP to produce a novel amorphous calcium fluoride phosphate (ACFP) phase of apparent composition $Ca_8(PO_4)_5F.xH_2O$[24]. The identification of this novel amorphous calcium fluoride phosphate (ACFP) phase led to the speculation that the formation of this phase is responsible for the observed additive anticariogenic effect of CPP-ACP and F. The anticariogenic mechanism of fluoride is now proposed to be the localisation of the fluoride ion at the tooth surface, particularly in plaque in the presence of Ca and phosphate ions[25]. This localisation increases the degree of saturation with respect to fluorapatite (FA) thus promoting remineralization of enamel with FA[25]. It is clear that for the formation of FA [$Ca_{10}(PO_4)_6F_2$], calcium and phosphate ions must be present with the fluoride ion. The additive anticariogenic effect of CPP-ACP and F may therefore be attributable to the localisation of ACFP at the tooth surface by the CPP which in effect would co-localise Ca, Pi and F. This hypothesis was tested in a mouthwash study where the ability of a 4.0% CPP-ACFP mouthwash used thrice daily to increase supragingival plaque calcium, inorganic phosphate and fluoride ions was determined[24]. The 4.0% CPP-ACFP solution used as a mouthwash contained 176 mM calcium ions, 128 mM phosphate ions and 18 mM (342 ppm) F ions. The use of the mouthwash resulted in a 1.9 fold increase in plaque calcium to 336 ± 107 µmol/g dry wt of plaque; a 1.5 fold increase in plaque phosphate to 471 ± 113 µmol/g dry wt of plaque; and a 3 fold increase in plaque fluoride ion to 43.77 ± 45.70 µmol/g dry wt of plaque. Although these marked increases in plaque calcium, phosphate and fluoride were found, calculus was not observed in any of the seventeen subjects, suggesting that the plaque calcium fluoride phosphate remained stabilised as the amorphous phase by the CPP and did not transform into a crystalline phase. These increases in the supragingival plaque levels of Ca, phosphate and fluoride ions are markedly greater than those obtained in a similar study with toothpastes containing 1000ppm F (MFP and NaF) used twice daily for a similar time period[26]. These results suggest that the CPP are an excellent delivery vehicle to co-localise Ca, fluoride and phosphate at the tooth surface in a slow release amorphous form with superior clinical efficacy. Recently, a dentifrice formulation containing 2% CPP-ACP plus 1100 ppm F was shown to be superior to a dentifrice containing only 1100 ppm F in remineralisation of enamel subsurface lesions with mineral that was more resistant to acid challenge (Reynolds *et al*., unpublished).

Commercial development of CPP-ACP

The CPP-ACP technology has been commercially developed by Recaldent Pty Ltd and is now available in sugar-free chewing gum and in a dental professional product called Tooth Mousse™ manufactured by GC. The Tooth Mousse product contains 10% w/w CPP-ACP and this product has been used clinically for the treatment of mild to moderate fluorotic lesions and for the reversal of white spots after orthodontic debanding.

Conclusion

The anticariogenicity of CPP-ACP has been demonstrated in the rat caries model, *in vitro* remineralisation/demineralisation models and human *in situ* clinical trials. The proposed anticariogenic mechanism of the CPP-ACP is the localisation of ACP at the tooth surface, which buffers the free calcium and phosphate ion activities, thereby helping to maintain a state of supersaturation with respect to tooth enamel preventing enamel demineralisation and enhancing remineralisation. Oral care products containing CPP-ACP (Recaldent™) are now commercially available in Australia, USA, Europe and Japan.

References

1. Loesche W. Role of *Streptococcus mutans in human dental decay*. Microbiol Revs 1986; 50:353-380.
2. Armfield J, Roberts-Thomson K, Spencer A. *Australia's Health 2000: the seventh biennial health report of the Australian Institute of Health & Welfare. Canberra*. 2000.
3. Crowley S, Antioch K, Crater R, Conway L, Matheis C. *The Economic Burden of Diet-related disease in Australia. Paper prepared for the Nutritional Food and Nutrition*. Centre for Health Program Evaluation and the Aust Inst Health. 1992.
4. Spencer A, Wright F, Brown L, Brown L. *Changing caries experience and risk factors in five- and six-year-old Melbourne children*. Aust Dent J 1986:160-165.
5. Reynolds EC. *Anticariogenic complexes of amorphous calcium phosphate stabilised by casein phosphopeptides. A review*. J Spec Care Dent 1998; 18:8-16.
6. Reynolds E, Johnson I. *Effect of milk on caries incidence and bacterial composition of dental plaque in the rat*. Arch Oral Biol 1981; 26:445-451.
7. Reynolds EC, del Rio A. *Effect of casein and whey-protein solutions on caries experience and feeding patterns of the rat*. Arch Oral Biol 1984; 29:927-33.
8. Reynolds EC, Black CL. *Reduction of choclate's cariogenicity by supplementation with sodium caseinate*. Caries Res 1987; 21:445-51.
9. Reynolds EC, Black CL. *Confectionery composition and rat caries*. Caries Res 1987; 21:538-45.
10. Reynolds E. *The prevention of sub-surface demineralisation of bovine enamel and change in plaque composition by casein in an intra-oral model*. J Dent Res 1987; 26:1120-1127.
11. Reynolds E, Black C. *Cariogenicity of a confection supplemented with sodium caseinate at a palatable level*. Caries Res 1989; 23:368-370.
12. Reeves R, Latour N. *Calcium phosphate sequestering phosphopeptide from casein*. Science 1958; 128:472.
13. Swaisgood H. *Chemistry of milk proteins*. In: Fox P, ed. Developments in Dairy Chemistry-1. London: Applied Science Publishers, 1982.
14. Ametani A, Kaminogawa S, Shimizu M, Yamauchi K. *Rapid screening of antigenically reactive fragments of alpha s1-casein using HPLC and ELISA*. J Biochem (Tokyo) 1987; 102:421-5.
15. Reynolds E. *Production of Phosphopeptides. Patent Application PK5706*. 1992.
16. Reynolds E, Riley P, Adamson N. *A selective precipitation procedure for the purification of multiple-phosphoseryl containing peptides and their identification*. Anal. Biochem 1994; 217:277-284.
17. Reynolds E, Cain C, Webber F, Black C, Riley P, Johnson I, Perich J. *Anticariogenicity of tryptic casein- and synthetic-phosphopeptides in the rat*. J Dent Res 1995; 74:1272-1279.
18. Reynolds E. *Anticariogenic Phosphpeptides. US Patent No 5015628*. 1991.
19. Reynolds EC, Black CL, Cai F, Cross KJ, Eakins D, Huq NL, Morgan MV, Nowicki A, Perich JW, Riley PF, Shen P, Talbo G, Webber FW. *Advances in enamel remineralisation: anticariogenic casein phosphopeptide – amorphous calcium phosphate*. J Clin Dent 1999; X:86-88.
20. Shen P, Cai F, Nowicki A, Vincent J, Reynolds EC. *Remineralisation of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate*. J Dent Res 2001; 80:2066-70.
21. Reynolds EC, Cai F, Shen P, Walker GD. *Retention in plaque and remineralisation of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum*. J Dent Res 2003; 82:206-11.
22. Iijima Y, Cai F, Shen P, Walker G, Reynolds C, Reynolds EC. *Acid resistance of enamel subsurface lesions remineralised by a sugar-free chewing gum containing casein phosphopeptide – amorphous calcium phosphate (CPP-ACP)*. Caries Research 2004; In press.
23. Reynolds EC. *Remineralisation of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions*. J Dent Res 1997; 76:1587-95.
24. Reynolds E. *Calcium Phosphphopeptide Complexes. International Patent Application No. PCT/AU98/0016*. 1998.
25. Thylstrup A, Fejerskov O. *Textbook of Cariology*. Munksgaard, 1986.
26. Sidi A. *Effect of brushing with fluoride toothpastes on the fluoride, calcium and inorganic phosphorus concentrations in approximal plaque of young adults*. Caries Res 1989; 23:268-271.

Getting the Fluoride Balance Right: Children in Long Term Fluoridated Communities

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Presented at 14th Biennial Conference of the Australian and New Zealand Society of Paediatric Dentistry, 20 March 2004, Melbourne

This presentation will focus on considerations in the fluoride balance for children in long term fluoridated communities, where the issue of 'balance' addresses the need for maximum reduction in dental caries while at the same time avoiding the possibility of dental fluorosis. The current status of community water fluoridation in Australia, and the current patterns of dental caries in children will be summarised, followed by a consideration of fluorosis and the discretionary sources of fluoride available for children today. Finally, the question of whether fluoride supplements, topical gels, rinses and varnishes still have a place in long term fluoridated communities will be discussed, and ways to maximize the effects of fluoride toothpastes will be considered.

Where is community water fluoridation up to in Australia?

Community water fluoridation (CWF) is still one of the best public health bargains available, costing Australians approximately 50 cents per person per year. Dental Health Services Victoria (DHSV) estimates that over $1 billion has been saved in dental expenses in the last 25 years in Melbourne, since the introduction of CWF.

There have been some notable anniversaries of CWF in Australian communities over the past three years, with Beaconsfield, Tasmania leading the way with a 50th anniversary, followed by Canberra and Hobart (40th), Sydney and Perth (35th), Adelaide and Darwin (31st), and Melbourne (25th). The notable exception among this list of major Australian cities is Brisbane, where the water remains non-fluoridated. Approximately 65% of the Australian population now live in areas supplied with fluoridated community water. Nonetheless, new proposals for the introduction of CWF in non-fluoridated rural cities and towns still face strong anti-fluoridation action with media sensationalism often highlighting minimal risk ahead of overwhelming dental benefits.

Twenty-five years later…new caries patterns in children and adolescents

As a result, caries patterns have changed dramatically in Australian children and adolescents. Dental Health Services Victoria reports that the average six-year old child in non-fluoridated Victoria has 40% more decayed teeth than the average six-year old in fluoridated Victoria. A recent report on 4-6 year olds in North Brisbane, Queensland reported that the mean dmft had fallen from 2.4 in 1988, to 1.7 in 1997, and to 1.4 in 2001; the distribution of caries-free children in this age group increased from 45.9% in 1988 to 66.3% in 2001 (Hallett and O'Rourke, 2002). The decline in caries prevalence and severity was accompanied by slower lesion progression. By 2001, caries in this age group was associated particularly with children of non-Caucasian and non-English speaking background and of low socio-economic status (Hallett and O'Rourke, 2002).

Despite such declines, the prevalence of early childhood caries (ECC) appears to be unabated and has not been affected by the caries reductions experienced by other age groups. The correlates of ECC are now becoming apparent. In particular, ECC appears to be predisposed by enamel defects of the primary dentition. A study in Brisbane of toddlers found significantly higher prevalence of enamel hypoplasia of the primary dentition in very low birth weight children, and a significant association between enamel defects and caries in this group (Lai *et al*, 1997). Statistical models for ECC based on a large set of variables studied in 2,515 children aged 4-6 years in Brisbane implicated four major variables in the final model, as follows: low family income, sleeping with a nursing bottle, sipping from a nursing bottle and ethnicity other than Caucasian (Hallett and O'Rourke, 2003).

Great gains from CWF have been achieved among adolescents since 1955, when the mean dmft/DMFT for 12 year-olds was 9.3 and only 1% was caries-free. The mean dmft/DMFT scores for this age group declined to 1.4 (in 1990), to 0.9 (1996), and to 0.8 in 1998 when 54% were caries-free (Armfield *et al*, 2001). By 2001, most caries was located in pits and fissures, and concentrated in a few children (Armfield *et al*, 2001).

Recent caries increases seen nationally in children

More recently, there has been a disturbing increase in caries, noted nationally in 5-6 year-olds over the period 1996 to 1999 (Armfield, 2003). Australia-wide data collected by the Dental Statistics Research Unit at the University of Adelaide and reported in 2003, have described increases in decayed teeth of 22% among 5 year-olds and of 8% among six year olds, and indicate the caries greatest experience is now concentrated in 10% of six year-olds, where the dmft approximates a score of 8 (Armfield, 2003). Such observations have led to several speculations to explain the recent increases, suggesting that fluoridated communities may be becoming complacent to the problem, or the profession may have over-reduced exposure to fluoride by topical measures, or the community may be consuming too much non-fluoridated water such as from bottled sources. To this point, there is no evidence to substantiate any of these speculations, but each is worthy of consideration.

Bottled water and home water filters

Over the past two decades, there has been an exponential market growth in bottled water sales. Marketed as pure,

healthy and clean, bottled water has become a primary source of hydration for an increasing proportion of the younger population, and increasing consumption of water instead of other beverages is consistent with sound nutritional advice. The standards for bottled water for microbiological and inorganic contents and for manufacture are controlled by the Australian and New Zealand Food Association and the Australian Bottled Water Institute, respectively (Bergman, 2003). The requirements include labeling of compounds of 5mg/L or more (such as sodium, potassium, bicarbonate, chloride and magnesium), and fluoride (labeling not required) should not exceed 1.7mg/L (Bergman, 2003). Information on the fluoride content of bottled water products is not readily available to the consumer (eg. via manufacturer hotlines), and while about 20 manufacturers in the USA add fluoride to their bottled water there is no evidence that such is occurring in Australia (Bergman, 2003). A recent study of the fluoride and microbial contents of ten brands of bottled water available in local supermarkets in Melbourne found that all contained less than 0.1 mgF/L (ie. less than 1ppmF) and less than 1CFU/mL. For comparison, Melbourne city water was measured at 1mgF/L (1ppmF), and the microbiological standard for reticulated water is less than 100CFU/mL (Cochrane *et al*, 2002).

It is speculated that consumers using bottled water in fluoridated communities *may* be losing out on the benefits of fluoride, but there is no evidence so far for the relative contribution of bottled water. It is recommended that patients be encouraged to drink fluoridated water and that patients with high caries rates be discouraged from using bottled water. The profession should take an active part in encouraging manufacturers to label the fluoride content on bottled water products.

Many patients request information on the effect of home water filters on the fluoride content of reticulated water. The ADA Practical Guidelines for 2000 indicates that ion exchange filters, reverse action (osmosis) filters, and water distillers can all remove fluoride from domestic water supplies, while carbon filters and ceramic filters do not remove fluoride (ADA Practical Guidelines, 2000).

Fluorosis and discretionary sources of fluoride for children

The appropriate daily fluoride intake in children should maximise the prevention of caries while limiting the risk of dental fluorosis. Today, the discretionary fluoride sources for children are multiple: diet, toothpastes, topical applications and supplements. Those posing possible risk factors for fluorosis include infant formulas, ingested fluoride toothpastes and fluoride supplements (Evans and Stamm, 1991). It is recommended that fluorosis on a community basis should be controlled by reducing the discretionary sources of fluoride rather than be reducing or removing CWF (Burt, 1992; ADA, Feb 2003).

The effects of fluoride on enamel are cumulative, rather than a specific threshold dose and depend on total fluoride intake from *all* sources and duration of exposure. Mild fluorosis can occur with ingestion of 2mgF or more per day, especially in fluoridated communities (Burt, 1992). The lower boundary of fluoride intake which can result in mild or very mild fluorosis has been estimated to be 0.04-0.07mgF/kg body weight/day in children, which is equivalent to 0.4-0.7mgF/day for a child weighing 10kg (Burt, 1992).

The fluoride intake for a child at age 22-36 months is of significance for anterior fluorosis, as ameloblasts exhibit a particular susceptibility to fluoride-induced change during the early maturation stage of enamel. Enamel mineralisation appears uniquely sensitive to free fluoride ions where the ion affects the breakdown and withdrawal of enamel matrix proteins. An excellent review of the pathogenesis of fluorosis has recently been published and the reader is directed to this for an in-depth discussion of current concepts (Aoba and Fejerskov, 2002).

Is there still a place for fluoride supplements?

In 1992, the Discretionary Fluoride Committee of the National Health and Medical Research Council (NHMRC) of Australia proposed the daily fluoride supplement schedule shown below *(Table 1)*.

This schedule was developed for high risk patients living in areas of <0.5mgF/L. The fluoride level of domestic water supplies in Victoria can be checked by consulting the 'Guide to Fluoride Levels in Water Supplied to Victorian Towns and Cities', published by the Department of Human Services, Victoria, 1993. Noting that the maxillary incisors are most susceptible to fluorosis at age 2-3 years, the dose does not increase beyond 0.25mg until age 4 years. The schedule has no upper limit, recognising that high caries risk may continue beyond age 8 years (Riordan, 1996). Tablets are typically marketed as 1mgF (as a 2.2mg NaF tablet containing 1mgF and 1.2mgNa), 0.5mgF (as a 1.1mg NaF tablet containing 0.5mgF and 0.6mgNa), and 0.25mgF (as a 0.55mg NaF tablet containing 0.25mgF and 0.3mgNa).

It was recommended that the supplements should be formulated as lozenges rather than tablets, and that the lozenges should be chewed and swished before swallowing in order to achieve a topical as well as systemic benefit. Since a supplement gives a peak plasma level (proportional to the dose), in about 20 minutes after ingestion, it was recommended that for a daily dose of 0.5mgF, one tablet of 0.25mgF should be taken twice per day in order to obtain maximum benefit without steep plasma peaks (Riordan, 1996). Of interest, this dose schedule was never officially ratified by the NHMRC, but it has been widely implemented by health departments and dental associations throughout Australia.

Table 1: Proposed Daily Fluoride Supplement Schedule

(Discretionary Fluoride Committee, NHMRC, Australia, 1992)

Age interval	Domestic water fluoride concentration <0.3mg/L	Domestic water fluoride concentration 0.3-0.5mg/L*
6 months to <4 years	0.25mg	0
4-8 years	0.5mg	0.25mg
8 years and over	1.0mg	0.5mg

** no supplement over 0.5mgF/L*

Recognising that a supplement chewed and swallowed does not provide the steady physiological levels of fluoride achievable with water fluoridation, many clinicians recommend instead dissolving the tablet in water (dissolving one 2.2mg NaF tablet in 1 litre of water gives 1ppmF water), and using this for domestic consumption.

In 1999, the Fluoridation Working Party of the NHMRC advised a lower dose draft schedule as shown below *(Table 2)* and published in the ADA Practical Guidelines in 2000.

The Fluoridation Working Party was disbanded by the NHMRC in 2001, citing insufficient funding to complete the work, and the revised draft schedule was not endorsed by the NHMRC (ADA News Bulletin, May 2003). This lack of national policy leaves fluoride counseling to dentists, and the NHMRC proposed dose schedule remains the current unofficial guideline in 2004 (Bergman, 2003).

In prescribing fluoride supplements, it is recognised that compliance is very variable, and is likely to reflect other preventive behaviours. The efficacy of supplements in caries prevention has been challenged as dubious, and there is convincing evidence linking fluorosis with supplementation. In a 1990 survey in Perth, Western Australia, Riordan found fluorosis in 40% children (Riordan 2002). In a follow-up survey in 2000 after supplement usage was reduced, a Thylstrup-Fejerskov score of zero was found to be much more prevalent, and the caries experience was in fact further reduced (DMFT down to 0.28 from 0.84 in 1990) (Riordan, 2002).

Is a fluoride supplement needed?

Due to national distribution of foods and beverages in Australia, products manufactured in fluoridated communities are now transported widely such that so-called non-fluoridated areas are actually receiving some benefit of fluoride, and are now considered to be in the 'halo' of fluoridated areas. Consequently, there may be few indications for blanket coverage with fluoride supplements in non-fluoridated areas. Fluoride supplementation is now an individual decision made by the dentist for high risk children, taking a number of factors into consideration.

This decision is made based upon the caries risk (dentist's judgement based upon experience is still the best indicator), age of the child, and the potential other sources of fluoride intake and utilisation. The clinician should take a brief inventory of fluoride sources (Messer and Mekertichian, 2003). For an infant, the parent could be questioned concerning the use of formula (soy-based or milk-based), any cultural habits and feeding practices, breast feeding (exclusively or partly), baby foods (commercial or home-made), and water sources (reticulated, tank, bore, bottled). For a preschooler, the parent could be questioned concerning water sources, cultural habits and feeding practices, use of fluoride supplements, fluoride topicals, current medications and toothpaste type (Messer and Mekertichian, 2003).

Is there still a place for professional topical gel applications?

In long-term fluoridated communities, a key question arises: Can children with low caries experience and using a fluoride toothpaste still benefit from a professional topical gel application? The answer to this can only be speculative, although anecdotal reports suggest a diminished benefit on a population basis. There appears to be no evidence-based data on the caries-reducing effect of gels in low caries children, and the only evidence comes as extrapolations from higher caries populations in the US where gel applications have been very useful in moderate to high risk patients (Bawden, 1992; Meskin, 1995; Clarkson, 1996). A recent report on 773 children aged 4-5 years old, resident in a low fluoride community in The Netherlands, followed for four years to age 8-9 years old with twice per year topical gel applications in comparison with a placebo gel, showed statistically significant caries reductions in first permanent molars but had no effect on primary molars (van Rijkom *et al*, 2004). However, the caries reductions were too low to be of clinical significance (van Rijkom *et al*, 2004).

Choosing a professional topical gel

Products available in Australia include 1.23% NaF neutral pH gel (12,300 ppmF), 1.23% APF gel (12,300 ppmF), and 0.4% SnF_2 gel (1,000 ppmF). As indicated, the 1.23% products are very high in fluoride concentration, and these should be limited to in-office use and are not for home use. Both NaF and APF deposit fluoride on surface enamel and in white spot lesions as CaF_2, which serves as a fluoride reservoir for remineralisation. Both compounds are probably equally effective in caries reduction. Due to its low pH, APF should be avoided on porcelain, composite and glass ionomer restorations, and also in those patients suffering from erosion and xerostomia.

The 0.4% SnF_2 gel (marketed as a treatment gel) contains 3,000 ppmSn and 1,000 ppmF, which is similar in fluoride composition to an adult fluoride toothpaste. Anecdotal reports from pediatric dentists indicate this product can promote remineralisation of lesions (such as in ECC cases) and reduce lesion severity, resulting in demonstrable surface hardening of demineralised enamel, prior to placing definitive restorations. The gel is applied to carious lesions as one drop on a cotton bud applied by the parent (who is told to 'paint on like nail polish'), several times per week after brushing and flossing. The parent must be very responsible and fully compliant in order to be delegated the responsibility as regular swallowing of this product in this age group could contribute to anterior fluorosis.

The question is asked frequently: Is a prophylaxis needed before a topical gel application? The answer is *not always*,

Table 2: Revised Daily Fluoride Supplement Schedule

(Fluoridation Working Party, NHMRC, Australia, 1999)

Age interval	Domestic water fluoride concentration < 0.3mg/L	Domestic water fluoride concentration > 0.3mg/L
0-3 years	0	0
3-6 years	0.5mg	0
6-16 years	1.0mg	0

as plaque is a reservoir for fluoride and serves as a recycling store. However, if a prophylaxis is done in order to remove stain or debris, or to teach oral hygiene, then a topical gel application is indicated because the prophylaxis removes some of the fluoride-rich surface enamel which should be replaced. Since fluoride is more effective on smooth surfaces than on occlusal surfaces, it is important to ensure the fluoride reaches the buccal and lingual surfaces (finger-mould the application trays to dried teeth), and the proximal surfaces (floss the teeth after gel application to drag the gel onto the proximal surfaces). Avoid swallowing of excess fluoride gel by placing only three 'corn kernels' of gel in each tray (total of less than 1mL), then flow these together with a cotton bud; maintain suction during the four minute application time and encourage the child to 'make a kiss and spit into the straw' (saliva ejector) at the end to remove all the fluoride-rich saliva.

Is there still a place for home rinses?

Supervised fluoride rinse programs can reduce caries by 20-50% in children. Daily and weekly rinses are available, marketed typically in Australia as 0.05% NaF (227 ppmF) and 0.2% NaF (910 ppmF), respectively. Due to risks associated with swallowing, these should only be used in children after the eruption of the permanent incisors. Such products can be of particular use for children who are unable to brush, those at high caries risk (eg. post irradiation xerostomia), or in times of increased caries susceptibility (eg. during orthodontic treatment).

New possibilities exist for the incorporation of fluoride into new caries-preventive modalities containing casein phosphopeptide-amorphous calcium phosphate (CPP-ACP). This casein derivative with anti-cariogenic properties was developed in the laboratory of Professor E Reynolds at the University of Melbourne School of Dental Science (Reynolds, 2000) is currently marketed as a tooth creme (Tooth Mousse™, GC Corporation, Japan), and as a therapeutic chewing gum (Recaldent™, GC Corporation, Japan). Initial laboratory studies and clinical trials on the inclusion of fluoride show promising results (Reynolds *et al,* 2002).

Is there still a place for concentrated varnishes?

One concentrated varnish (Duraphat™, Colgate Oral Care) currently widely used in Australia is formulated as an alcoholic solution of natural varnishes containing 50mgNaF/mL (as 5% NaF, 2.26% F, 22,600 ppmF). This binds to enamel and also plaque acts as a reservoir, slowly releasing fluoride over a period of 12-48 hours.

"In order to avoid fluorosis...children should use a pediatric toothpaste until after the eruption of the permanent incisors."

Due to the slow release and high concentration of fluoride, varnishes have the potential to cause acute toxicity and careful case selection is essential. Used only after the eruption of the permanent incisors, the varnish is useful in hypersensitive areas, on newly erupted teeth, in high-risk patients and those in high risk categories, and for local remineralisation of white spot lesions.

Can the effects of fluoride toothpastes be maximised?

Children's toothpastes marketed in Australia currently contain 400 or 500 ppmF, and adult toothpastes contain 1,000 and 1,500 ppmF. In order to avoid fluorosis from regular swallowing of adult strength toothpastes, children should use a pediatric toothpaste until after the eruption of the permanent incisors. Toothpastes containing either NaF or NaMFP appear to be equally effective, and both achieve about 30% caries reduction with regular use. With reference to fluoride concentration, a 2003 evidence-based report based on a meta analysis of 7 randomised controlled trials and examining the caries experience in permanent teeth has indicated that 1,000 ppmF toothpastes are significantly more effective than 250 ppmF toothpastes. There was insufficient data to draw any conclusions concerning the relative efficacy of 500ppmF toothpastes (Ammari *et al,* 2003).

In choosing a pediatric toothpaste, parents should be encouraged to read the labels and told what to look for with reference to fluoride concentration, as several manufacturers package adult strength toothpastes in tubes decorated with children's motifs. Since up to 30% of the toothpaste on the child's brush can be swallowed, parents should brush and floss for their child until about 7 or 8 years of age, and should supervise 'play brushing'. A pea-size amount of low fluoride toothpaste should be smeared sideways across the brush head.

With particular reference to preschoolers, brushing twice per day with a fluoride toothpaste for under two-year olds has been shown to significantly reduce caries, and teaching preschoolers to 'swish and pump' a fluoride toothpaste between the primary teeth can reduce caries by 30%. Two large evidence-based studies concerning the effectiveness of fluoride toothpastes in children have been conducted recently. One report assessed 70 'high quality' studies involving 42,500 children, and the second report assessed 54 such studies (Marinho *et al,* 2003; Twetman *et al,* 2003). Both performed meta analyses on the assembled data, and both concluded that the effect of a fluoride toothpaste in children was increased by supervised brushing, a higher baseline DMFS, a higher fluoride concentration, and a higher frequency of use. Of interest, the effect was unaffected by exposure to community water fluoridation (Marinho *et al,* 2003; Twetman et al, 2003).

Conclusions

Clearly there are new challenges today in achieving the correct balance for fluoride utilisation in long-term fluoridated communities, and there is no room for complacency in terms of caries reduction. Fluoride plus other traditional measures may not be enough for the very high caries risk individual. All fluoride products need careful supervision, both in the dental practice and in the home. Combinations of casein-phosphopeptide-amorphous calcium phosphate and fluoride may open up new possibilities for preventive products suitable for professional and home delivery.

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(Author's note: A complete reference list is available from the author).

ANZSPD Federal Secretary-Manager's Report

1. Colgate *Bright Smiles, Bright Futures* Award.

I have received notification of this award from the International Association of Paediatric Dentistry. This is a world-wide award programme, aimed at stimulating the development of innovative preventive oral health programmes for children. It offers a prize of US$2,500 and economy airfares to the Sydney IAPD Congress in November 2005 to any individual or organisation able to create or implement an oral health community programme. These may be in any academic, clinical or community-based setting which serves children, such as a school, health or community centre. Ideally, the organisers would like the programme to be underway now, so that it can be demonstrating results by 2005.

The Award is being conducted out of the Colgate offices in New Jersey, U.S.A. and applications needed to be received by 1 March 2005.

Included with the notification was an official award application form. I can copy this form and send it to anybody or group in Australia or New Zealand who may be interested.

Otherwise, further information can be obtained from Kathryn Parrish of Colgate. Her email address is kathryn_parrish@colpal.com.

2. Federal Council of A.N.Z.S.P.D.

The Federal Council of A.N.Z.S.P.D. is planning to meet at the time of the 31st A.D.A. Congress in Adelaide, South Australia, 3-7 March 2005. I shall be contacting branches closer to the time seeking any items for inclusion on the agenda. In the meantime, branches and individuals may wish to give consideration to any matter they would like discussed.

3. The I.A.P.D. Congress in Sydney

This is getting closer. As had been resolved by the Federal A.N.Z.S.P.D., a AUD$50,000.00 donation has been made to the Congress Organising Committee, specifically to be used towards paying for the venue for the Congress, the Sydney Convention and Exhibition Centre. This cheque was forwarded in November.

4. A.N.Z.S.P.D. Essay Competitions

The Under-graduate Essay Competition is being judged as this report is being compiled. The Post-graduate Essay Competition has been judged, and the Judges have recommended that the prize not be awarded for 2004.

5. The Royal Australasian College of Dental Surgeons Inc.

has a Board of Studies for Paediatric Dentistry, and over the years, the College has asked A.N.Z.S.P.D. to submit a list of nominees from which they would appoint some of the four members to the Board of Studies. This request has been made again, but after some consultation, the Executive has decided it is probably more appropriate for such a list to be submitted by the Australasian Academy of Paediatric Dentistry instead of A.N.Z.S.P.D.

Alistair Devlin

Secretary-Manager

ANZSPD – Branch News 2004

New Zealand

Dr Bernadette Drummond has been appointed Associate Professor at Otago University. Bernadette has provided significant input to the NZ branch of ANZSPD and also to paediatric dental services in New Zealand. She has mentored many along the way for which we are all very grateful. We congratulate Bernadette on her recent appointment.

Dental health week was held early in 2004 with a significant slant being the plight of children's teeth in New Zealand. Significant input was made by NZDA especially David Crum, NZDA's executive director, Callum Durward and Nina Vasan. It was a multimedia exercise with several newspaper articles and TV coverage.

The NZDA conference was held in (a very cold) Christchurch om September 2004 with some very good speakers with paedo interest – Prof. Stanley Malammed on local analgesia with particular emphasis on the dangers of LA and children, Dr A Andreason with a comprehensive review of trauma management. Dr Bernadette Drummond's and Dr Nina Vasan's lectures on paediatric dentistry also received good support and feedback.

The executive is finalising some courses for general practitioners for this year which we are yet to advertise.

MaryAnne Costelloe

ANZSPD – Branch News 2004

New South Wales

The NSW Branch had a very busy year in 2004.

We had two well attended dinner meetings in the second half of the year. In August we had a mixture of topics. Dr Kotala spoke on the state of dentistry and the changes he has seen over the last three years in Laos. Drs Marks, Hibbert and Mekertichian gave amusing and thought provoking talks on 'resurrection of clinical failures'. In October we were very pleased to have Dr Callum Durward as our invited international speaker for our final meeting. As a number of members of ANZSPD have visited Cambodia as part of ongoing dental teaching, Dr Durward spoke on the success and failure of oral health services for children in Cambodia. After seeing what important work both Drs Kotala and Durward are doing in these disadvantaged countries, the NSW ANZSPD branch hopes to be able to provide financial and materials support to these regions.

At our annual general meeting in October a new executive was unanimously voted in. Dr Soni Stephen and Dr Erin Mahoney are stepping down as President and Secretary respectively. Dr Sally Hibbert is our new branch President and Dr Philippa Sawyer is the new branch Secretary. Thankfully Dr Anthony Burges is staying on as treasurer.

Drs Michael Malandris and Juliette Scott have recently finished their MDSc as specialists in Paediatric Dentistry at the University of Sydney and we wish them well in the future. Michael is off to Adelaide and Juliette is staying in Sydney to get married to our branch treasurer in March! Dr Erin Mahoney is off for 18 months to Canada but will be back in Sydney for IAPD 2005. The organising committee for IAPD Sydney 2005 is meeting regularly. The registration brochure is now finalised. You can register and submit an abstract at www.iapd2005.com or email: Rebecca.deal@icmsaust.com.au. We look forward to seeing everyone in Sydney!

Erin Mahoney

Western Australia

The Annual Mid Winter meeting of the WA Branch was held on Saturday, 31 July 2004. This year, the Branch chose to return to the south west of the state, specifically to the brand new Bunker Bay Quay West Resort. This Resort, which is located at the northern gateway to the Margaret River wine-producing region, occupies a prized location on a north facing beachfront. Those in attendance were treated to an illuminating programme. Sam Gue from Adelaide spoke on the management of traumatic injuries to primary teeth and on oral soft tissue pathology in children and adolescents; Sally Hibbert from Sydney gave an update on dentine caries and on the management of the avulsed tooth, in addition to speaking on the restoration of primary molars. As had been the case when Sally had visited Perth in 2003, she didn't shirk from being controversial, and her presentations provoked much discussion. Sally was actually 'first reserve' – originally, it was hoped Erin Mahoney could visit, but as an important event in her life drew close, Sally kindly stepped in. The final presenter for the day was Dr Blaise Johnson, who called upon her background in paediatric dentistry and psychology to speak on stress management – in this instance, for the operator. Probably, this would have been the first time most in the audience had been to a course where they were issued with towels, had the lights dimmed, listened to soothing music and then received a complimentary compact disc of other examples of such music! The customary after course dinner was held at Wise's Restaurant, which is located in a commanding position high up overlooking Geographe Bay. It was a fitting conclusion to a memorable day; the Branch is indeed indebted to Peter Readman who was responsible for organising the day.

The W.A. Branch has appointed a committee to organise the 15th A.N.Z.S.P.D. Convention. The organising committee has been busy already, even though the Convention is some time off yet. After canvassing opinion at the time of the 14th Convention in Melbourne, the decision has been made to hold the Convention in Broome, which is located in the Kimberley region in the far north of Western Australia. The chosen dates are 23-27 May 2007. The venue will be the famous Cable Beach Club Resort.

The University of Western Australia has appointed a full time Senior Lecturer in Paediatric Dentistry. Dr Boyen Huang arrived in Perth from overseas in November and has commenced at the School of Dentistry. All Branch members look forward to the opportunity of meeting Dr Huang. The Annual General Meeting of the Branch was held on 10 December. This was a meeting with a difference. It was held at the so-called 'Megazone', which is a Timezone type of games area for children located at the Princess Margaret Hospital for Children.

Alistair Devlin

Queensland

The branch had another active year in 2004 with regular meetings and lectures. At the February 2004 AGM meeting Dr Mathew Gentner, Endodontist, spoke on 'Dental Trauma – Fracture and Root Resorption'. Office Bearers were elected for 2004 – Dr Robin Smith as President, Dr John Rutar as Secretary/Treasurer, Dr Kerrod Hallett as Federal Representative and Dr Michael Kenwood as Committee Member.

In May, Dr Richard Roylance, Paediatrician, spoke to the members on the topic of 'Child Abuse and Neglect – Implications for Dental Practice'. Dr Kerrod Hallett spoke to the Society in August on the topic of 'Dental Appliances for Special Needs Children'.

Our Annual Clinic Day was held during November in Brisbane at the Diana Plaza Hotel. Our invited keynote speaker was Dr John Winters, Federal President ANZSPD, and he was supported by two invited guest speakers – Dr Kylie Pearce and Surita Meintjes. The lecture program included the following topics – Complete Oral Rehabilitation under General Anaesthesia, Digital Photography in Dentistry, Electrosurgically Modified Pulpotomy, Molar Incisor Hypo-

mineralisation – Aetiology and Management and Aspects of Nutrition and Tooth Wear. Members were given excellent presentations and all enjoyed the day.

At the November meeting dates were organised for the 2005 Branch meetings. Thanks are extended to Office Bearers whose tireless work throughout the year produced a successful year.

John Rutar

South Australia

The South Australian Branch had a number of meetings in 2004 that have covered various areas of interest in paediatric dentistry. Angus Cameron visited for a day course that was run in conjunction with the Post Graduate committee and members took the opportunity to hear Angus talk on a range of topics. There was good attendance at this course as it was well supported by local members and the School Dental Service. The committee has organised another clinical day next year focusing on management issues.

The evening meeting was to have been a continuation of discussions from the day, but unfortunately Angus was unable to attend as he had been struck down by a tummy bug. The evening went well none the less, with Dr John Burrow being awarded Life Membership for his contribution to Paediatric Dentistry. Following this meeting there was a joint meeting with the Oral Medicine and Oral Surgery Study Group. This proved very successful. The speaker was one of the Crows medical team. He discussed the dental and health aspects related to running an AFL team. One interesting point was the monitoring of the health and well being of the many volunteers, many of them falling into the over 65 years age group. So, not only the players need to be kept in peak form!

The final meeting for the year was a lecture on the orthodontic treatment of disabled children by a local orthodontist, Dr Damian Gallagher. Damian works in both private practice and at the Women's and Children's Hospital. He talked on the treatment challenges and options for children with disabilities.

This year we began our programme with our AGM and a visit from the Clown Doctors. We are looking forward to another busy year, the highlight of which will be the IAPD meeting in October.

Sue Springbett

Victoria

2004 has been a very busy year for the Victorian branch of ANZSPD. The year started officially in March with Melbourne hosting the 14th Biennial Conference. The preparations however were well underway by the end of 2003 and the committee, led by Mala Desai with Chris Olsen, Karen Kan, Jodie Heap, Felicity Wardlaw and John Sheahan are to be congratulated on a magnificent event. The conference was opened by the Hon Bronwyn Pike, the Victorian Health Minister but will be remembered as the occasion at which Bridget Sheahan made her debut performance at the age of 8 responding very professionally to the opening address. The keynote speaker was Dr Stephen Fayle from Leeds in the United Kingdom. Once everyone got use to his Northern English accent, Stephen provided us with hours of invaluable information. His presentations were polished and professional covering a wide spectrum of clinically relevant topics including molar-incisor-hypomineralisation, caries prevention and behaviour management. He introduced us to the Wand, a computerised method of delivering local anaesthesia painlessly and discussed the use of general anesthesia in paediatric dentistry. His contribution to the session on the use of fluorides in caries prevention highlighted the diversity of opinion and lack of consistency around the world with respect to fluoridation and fluoride protocols. Stephen was ably supported throughout the program by a broad cross section of local paediatric dentists including Bernadette Drummond, Louise Brearley-Messer, Peter Readman, Nicky Kilpatrick, Nina Vasan, Peter Wong, Angus Cameron and Margarita Silva. In addition non dental speakers further broadened our horizons with Professor Eric Reynolds reviewing the development of Casein Phosphopeptide – Amorphous Calcium Phosphate, Dr Rob McDougall providing an anaesthetist's perspective on our management of children and Tissa Jayasekera giving an orthodontic perspective to the management of compromised first permanent molars. As usual Melbourne put on a wonderful event including a gala dinner and many delegates were able to find time to pamper themselves with some high quality retail therapy. In short the 14th biennial conference was undoubtedly professionally, socially and financially successful.

As the conference occupied a significant amount of time in the early part of the year, the branch did not have an evening meeting until early June. Dr Igor Lavrin, an orthodontist laid out the issues surrounding the controversial 60 minutes program on the benefits and risks associated with orthodontic treatment. In a presentation of high quality, Igor unraveled some of the myths associated with orthodontic treatment and highlighted the inconsistencies associated with the media coverage including needless to say, what was not said. The audience left feeling more prepared to address the concerns of their patients. At this meeting the Des Crack prize was awarded to Dr Yvonne Chang.

Dr Michael Stubbs braved a Victorian winter's night to give an excellent presentation on oral medical problems in children. Michael is a specialist in oral medicine who also has an interest in Special Needs Dentistry. His presentation took the form of a number of clinical cases for which the audience where asked to provide diagnoses. He reminded us that one requires a logical approach to diagnosis so that we don't miss the less common pathologies.

The final lecture in our series was the Elsdon Story Memorial Lecture in honour of the late Professor Elsdon Story. Mrs Pat Storey and her family attended this lecture. This year Dr Luke Moloney, a specialist endodontist both in private practice and at the Royal Children's Hospital, discussed the management of traumatic injuries in young children. In particular he discussed the sequelae of avulsions and luxation injuries in the immature tooth. His talk supported the view that you can never really give up on a tooth; several of his cases appeared to have a hopeless prognosis but his longer term follow up radiographs demonstrated impressive retention of the tooth throughout the growth period.

At each of the evening meetings the dinner is preceded by a short

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The Little Bear who Sucked his Thumb

by Dr Dragan G Antolos

As a parent I feel ill-equipped and poorly trained in addressing the inevitable problems and dramas associated with raising two beautiful girls. What are the sterilisation guidelines for changing a 'pooey nappy'? How do I react when 'Jonathon' in Grade 6 gives my little girl a pillow in the shape of a love-heart (of all things) and tells her he likes her. Outside I'm laughing, inside I'm crying! The inevitable dramas make me shiver and sweat. At least I have all their dental problems covered. After all, I've been in general dental practice for twenty-two years, with a special interest in orthodontics. I can give them healthy teeth and a nice smile.

Anyway….. my eldest daughter Ellen decides to suck her thumb as an infant. No problem I say, I have this covered. 70% of infants will suck their thumb at some stage. Most will stop before they're two-years-old. And what a great pacifier that thumb is. She is the model baby, and very cute with the thumb and all.

Anyway….. Ellen turns three and she is still sucking her thumb. No problem I say. Most three to four-year-olds will give up before they are six. Her dad's a dentist – she will stop no problems.

Anyway….. Ellen turns six and she is still sucking her thumb. She develops an anterior open bite with an associated tongue thrust swallow pattern. How do you figure? Daughter of super-dentist, becomes a persistent thumb-sucker!

How can you tell which three-year-old will be a persistent thumb-sucker? I'm living proof that you can't. Which of those will create dental or developmental abnormalities? You may as well ask a fortune teller. From my experiences as a parent and dentist I have come to the following conclusions regarding thumb-sucking in children:

1. Thumb-sucking is acceptable up to three years of age (great pacifier!).
2. Between the ages of three and six, there is a window of opportunity to help stop the habit.
3. If the habit persists after eruption of permanent teeth, problems can be expected.
4. And most importantly, if the child does not have the **desire** to **stop**, all other attempts will fail. Any nagging or forceful pressure placed on a child will only make the problem worse.

So how do you start to address the problem in a child with a thumb-sucking habit without creating insecurities and anxiety?

Children love to collect books and have their parents read them stories. A colourful picture story book is an excellent format that kids understand and relate to. It is fun and non-threatening.

However, surprisingly, there is very little children's literature on the topic. Subsequently, I wrote and illustrated **The Little Bear who Sucked his Thumb**.

Oliver is a little bear with a thumb-sucking habit. Initially Oliver finds it comforting and fun, but soon decides it is time to stop his thumb-sucking. This proves to be more difficult than Oliver had first thought. So off he goes into the woods, to seek out a mystical dragon, who he is sure can help. The dragon shows Oliver how, with determination, and a little help, he can stop sucking his thumb.

The book can be purchased on the internet at **oliverthebear.com**, or by phoning **(03) 9850 4056** (during business hours). It is distributed by Pan Macmillan and can also be ordered through bookstores. The book retails at AU$24.95. Mail order will attract a $5.00 postage and handling fee.

It is a book that can be recommended to parents with children who suck their thumb, or as a useful book to have in the waiting room.

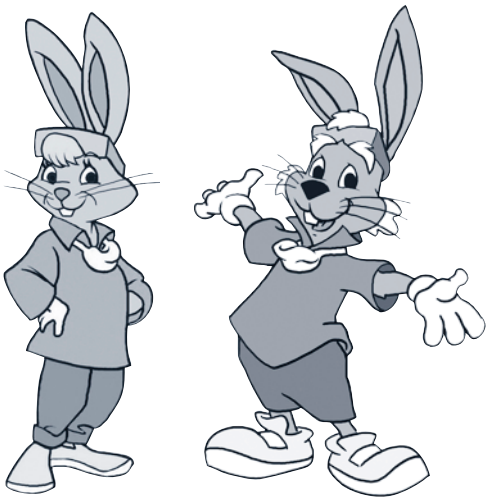
Colgate® Corner

by Dr Jackie Robinson
Colgate Professional
Relations Manager

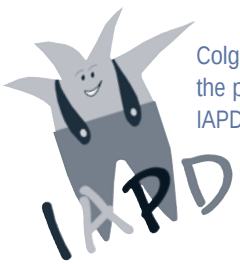
Colgate Bright Smiles Bright Futures

We are starting 2005 off with the launch of the Colgate Bright Smiles Bright Futures kits for Year 3 classrooms. The new kits contain the same high quality and variety of materials that you expect from the Colgate BSBF school education program:

- Dr Rabbit and the Legend of Tooth Kingdom video
- Brush for a Bright Smile poster
- Saving Tooth Kingdom from the Sorcerer Plakula poster
- Comprehensive teachers guide
- Student Take Home – My Adventures in Tooth Kingdom (32)
- Parent Take Home – Bright Smiles for the Family (32)
- Oral Health Message stickers (32)



The materials feature our oral health hero, Dr Rabbit, and introduce his new female counterpart, Dr Brushwell. The new graphics are fantastic! For more information and to order kits online, visit: www.colgateprofessional.com.au/BSBF



Colgate is very proud to be the principal sponsor of the IAPD Congress 2005

Changing faces in the Colgate team

Members of the Association from Queensland are undoubtedly aware that Narelle Bird and Barbara Whitaker have left Colgate. A new Colgate Territory Manager for Queensland, Hilary Berry, started in February and is working hard to meet everyone.

Narelle and Barbara will both be sorely missed within Colgate. Narelle was a Territory Manager for Colgate for 9 years; Barbara for 2 years. Both were well respected within Colgate and within the dental profession for their experience and expertise and for their professional approach. I know all of you join me in wishing Narelle and Barbara all the best.

Colgate product news

Colgate has lots of new products to talk about. We are particularly excited about the launch of the new 'Smiles' range of toothbrushes for children.

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Youth (Ages 5+)

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In New Zealand, orders for Colgate products are placed through Shalfoon Dental:

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The Colgate team of Territory Managers is here to assist you with the products you need in your surgeries.

Coming events

- **26-30 May 2005**

  58th American Academy of Pediatric Dentistry (AAPD) Annual Session

  Walt Disney World Dolphin, Orlando, FL

  www.aapd.org/events

- **31 October – 5 November 2005**

  20th International Association of Paediatric Dentistry (IAPD) Congress

  Sydney Convention & Exhibition Centre

  www.iapd2005.com

- **23-27 May 2007**

  15th Australian and New Zealand Society of Paediatric Dentistry (ANZSPD) Biennial Conference

  Cable Beach Club Resort, Broome WA

Victoria Branch News

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presentation given by one of the many postgraduate students in paediatric dentistry. Dr Siew Luan Toh interrupted her honeymoon to review the management of impacted incisors and Dr Caroline Howarth discussed tooth transposition. These short presentations have become a regular part of our dinner meetings and provide an opportunity for younger members to develop their presentation skills as well as updating us older members on clinical knowledge.

2005 will be a relatively quiet year for the Victorian Branch with the ADA congress in Adelaide and then the International Association of Paediatric Dentistry conference in Sydney in late October. The branch has made a significant financial contribution to the Sydney meeting which promises to be a highlight for all of us interested in improving the oral health of children. We certainly wish the NSW branch and in particular the IAPD organising committee all the very best and promise to be there in number to support the event.

Nicky Kilpatrick

AUSTRALIAN AND NEW ZEALAND SOCIETY OF PAEDIATRIC DENTISTRY

www.anzspd.org.au

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Submissions

All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD, or by email. Both PC and Mac formats are accepted. Media will not be returned. Address email to karenkan@optusnet.com.au. Please enclose your contact details and email address with all submissions.

Deadline next issue

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